CHEMICAL CARDIOVERSION OF SUPRAVENTRICULAR TACHYCARDIA WITH CALCIUM GLUCONATE

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Abstract—Background: Approximately 0.05% of Emergency Department visits in the United States are related to supraventricular tachycardia (SVT). The majority of patients convert with an atrioventricular nodal blocking medication. Case Report: We report a case of SVT that converted after administration of calcium gluconate after failing conversion with adenosine. Conclusion: Conversion to normal sinus rhythm resulted after administration of i.v. calcium in our patient. Additional investigations would be helpful in determining the causal vs. temporal association of conversion of SVT with calcium administration. © 2011 Elsevier Inc.

Keywords—supraventricular tachycardia; calcium gluconate; dysrhythmia; cardioversion

INTRODUCTION

Supraventricular tachycardia can be defined as any tachycardia initiated at the atrium or the atrioventricular (AV) node. Typically, it is a narrow-complex tachycardia with a regular, rapid rhythm. Atrial fibrillation and multifocal atrial tachycardia are two common exceptions. Rates vary from 150 to 250 beats/min, but are generally within 160 to 200 beats/min. Paroxysmal supraventricular tachycardia (PSVT) is an episodic presentation with a sudden onset and termination, usually occurring secondary to AV nodal re-entrant tachycardia. Epidemiologically, this rhythm disturbance occurs equally among races, twice as frequently in women, and increases with age (1,2). Frequently, it is found in those who are fatigued, and there seems to be a correlation with large consumption of coffee and alcohol, and cigarette use. There are an estimated 89,000 new cases per year and 570,000 persons with PSVT in the United States (1,2). Approximately 0.05% of ED visits in the United States between 1993 and 2003 were related to SVT.

Although PSVT is typically not life threatening, episodes should be treated or prevented. According to new American College of Cardiology/American Heart Association/European Society of Cardiology guidelines, Class I recommendations include vagal maneuvers, adenosine, verapamil, and diltiazem, in that order. Beta blockers, amiodarone, and digoxin fall under the Class IIb recommendations (3). Vagal maneuvers cause the carotid baroreceptors to sense an increase in blood pressure, which therein stimulates the parasympathetic nervous system to induce a reflex bradycardia. Adenosine, a Class V anti-dysrhythmic agent, causes a transient heart block and leads to successful conversion of PSVT in 90% of cases (4).

CASE REPORT

A 56-year-old African-American man presented to the Emergency Department (ED) complaining of heart palpitations and mild fatigue. He reported four previous similar episodes, most recently 1 year prior, and had been diagnosed with supraventricular tachycardia (SVT).
He stated that adenosine was used for conversion of his last three episodes, and vagal maneuvers successfully converted his second episode. He had no other medical problems, was not currently taking any medications, and stated that he was never prescribed medicine for his SVT. On initial presentation, he was a well-appearing man sitting comfortably in the stretcher, in no acute distress. His initial vital signs were: heart rate 160 beats/min, blood pressure 125/88 mm Hg, and SpO₂ 99% on room air. An electrocardiogram showed SVT (Figure 1).

He was treated following the SVT algorithm, with vagal maneuvers and carotid massage, which were both unsuccessful. He was then given adenosine 6 mg i.v. push, 12 mg i.v. push, and 12 mg i.v. push, without any change in rhythm. Before giving him diltiazem, we decided to pre-treat him with an amp of calcium gluconate because his blood pressure had dropped to 106/72 mm Hg during the prior unsuccessful conversion attempts. Approximately 5 s after administration of an amp of calcium i.v., his rhythm converted to sinus rhythm (Figure 2). His vital signs after conversion were: heart rate 86 beats/min, blood pressure 110/74 mm Hg, and SpO₂ 99% on room air. Per cardiology recommendations, he was placed on atenolol and followed-up as an outpatient. His post-conversion chemistry panel was within normal limits, including normal serum calcium levels. The patient did not keep his scheduled cardiology appointment at 4 weeks, and was lost to follow-up.

**DISCUSSION**

The algorithm for treatment of SVT begins with vagal maneuvers and carotid massage. Vagal maneuvers are efficacious in terminating about one-quarter of spontane-
ous SVT cases, and in one study there was no detectable difference in efficacy between the Valsalva maneuver and carotid sinus massage (5). When SVT is not terminated by vagal maneuvers, initial management involves intravenous adenosine. Adenosine is a short-acting drug that blocks AV node conduction; it terminates 90% of tachycardias due to AV nodal reentrant tachycardia (6). Typical adverse effects of adenosine include flushing, chest pain, and dizziness. These effects are temporary because adenosine has a very short half-life of 10–20 s.

After adenosine, calcium channel blockers (CCBs) are the typical alternative for SVT treatment, typically verapamil and diltiazem. Verapamil is a calcium channel blocker that also has AV blocking properties, a longer half-life than adenosine, and may help maintain sinus rhythm after the termination of SVT (7). Comparing the CCBs, verapamil led to successful conversion in 98.8% of patients, compared to 96.3% conversion in use of i.v. diltiazem (not statistically significant) (8). Similar to adenosine, CCBs can lead to flushing and headaches, and also may result in decreases in blood pressure. They are typically avoided when patients are hypotensive, or trending in that direction.

Calcium has been used in pre-treatment to prevent verapamil-induced hypotension in patients with SVT, decreasing the hypotensive effect of verapamil without compromising its anti-dysrhythmic effect (9). However, calcium has not been widely used for termination of SVT and has been suggested primarily for pre-treatment, not conversion of dysrhythmias.

O’Brien et al. described several cases of PSVT that converted to normal sinus rhythm within 1–2 min of receiving intravenous calcium salts, intended for pre-treatment in anticipation of verapamil therapy (10). The authors hypothesized that this was due to calcium’s effect on raising the blood pressure, leading to an increased cardiac parasympathetic tone, and directly slowing AV conduction. One additional case study reported that the use of i.v. calcium chloride as pre-treatment for verapamil administration for treatment of SVT led to ventricular fibrillation. In that case, however, the patient had an accelerated wide-complex tachycardia (11). In our case, we chose to give calcium before using diltiazem for pre-treatment purposes to avoid additional decreases in blood pressure. We were surprised by the immediate conversion after the calcium gluconate was administered.

CONCLUSIONS

Although not traditionally used in the conversion of SVT, i.v. calcium may have contributed to the successful conversion to normal sinus rhythm in our patient. As discussed above, this could be due to parasympathetic tone increases or its slowing effect on conduction at the AV node. Additional research in this particular area would be helpful to further assess calcium’s role, causal vs. temporal, in SVT treatment. Results of such work would help delineate calcium’s potential as an additional therapeutic option for SVT conversion or as a medication to be avoided due to its potential dangerous effects.

REFERENCES
